patients, the site of the leak may remain unclear or there may be a suspicion that there is more than one defect in the skull base. Under these circumstances a diagnostic or peroperative lumbar puncture using fluorescein dye will help to localise the area. The use of dilute fluorescein and a "pencil point" epidural needle has minimised the complications associated with this technique.[7]

Endoscopic closure has revolutionised the surgical management of CSF rhinorrhoea and has reduced the morbidity associated with it; a recent meta-analysis reviewed the use of endoscopic repair.[8] The sense of smell is almost always preserved using this technique but it is usually lost when a transcranial approach is used.[8] When endoscopic closure is used the length of time spent in hospital is usually restricted to 36 hours, and a craniotomy is avoided. Nasal endoscopic repair has a success rate of 90% at the first attempt and 97% after a second attempt.[8] Morbidity is minimal.

Although an intracranial approach has the advantage of allowing for the resection of any coexisting intracranial pathology, the success rates achieved using anterior craniotomy are less than 75%.[9 10] A frontal craniotomy often results in a loss of the sense of smell and uncommonly, but importantly, may be complicated by postoperative intracerebral haemorrhage, cerebral oedema, epilepsy, frontal lobe dysfunction with memory and concentration deficits, and osteomyelitis of the frontal bone flap. In addition, this technique requires the patient to spend five to seven days in hospital, results in hair loss along the incision line, and the patient must not drive until judged to have recovered from the operation. An extradural approach is, however, needed for defects of the posterior wall of the frontal sinus or defects larger than 5 cm because these cannot be managed endoscopically.

If a CSF leak occurs in conjunction with a tumour of the skull base or a severe fracture of the skull base, a craniotomy and removal of the posterior wall of the frontal sinus and its lining along with the anterior wall of the frontal sinus with split calvarial bone is indicated.[11] The use of a pericranial flap reduces the postoperative incidence of CSF leaks.

No prospective randomised study has been done comparing these techniques. However, on the basis of series reports[12–14] and a meta-analysis,[8] the differences in morbidity and success rates between the techniques make transnasal endoscopic repair the treatment of choice for most CSF leaks from the anterior cranium and sphenoid sinus.

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Depleted uranium and public health

Fifty years' study of occupational exposure provides little evidence of cancer

Depleted uranium, used in anti-tank weapons, provides a common thread that links concerns about leukaemia and other health effects in peacekeeping forces returned from the Balkans and worries about the environmental impact of debris from weapons in this war-weary segment of Europe. Unlike many agents that seem suddenly to prompt health concerns,[1] however, we know quite a lot about the health effects of depleted uranium.

Depleted uranium is derived from natural uranium mined from the earth's crust. Uranium is composed of three radioactive isotopes, U^{238}, U^{235}, and U^{234}, which decay to other radioactive elements and ultimately to stable non-radioactive isotopes of lead.[2–4] Uranium isotopes emit α particles during decay, which possess high energy but are poorly penetrating. Thus, uranium poses primarily an internal radiation hazard to tissue in close proximity.

Uranium is not very radioactive, owing to its isotopes' relatively long half lives (10^5-10^9 years). These compare with radon, a prominent member of the daughter progeny of the uranium decay cascade, which possesses a half life of 3.8 days and a radiological activity 10 000 times greater.[2–4] Depleted uranium possesses only 60% of the radioactivity of natural uranium, having been "depleted" of much of its most highly radioactive U^{234} and U^{235} isotopes.

Depleted uranium is also a heavy metal, like lead or arsenic, with toxicity a function of route of exposure, particle solubility, contact time, and rate of elimina-

BMJ 2001;322:123–4

tion.[2 5] Some of these chemical properties, high density, and tensile strength, made depleted uranium an attractive material for use in weapons.

Though its first combat use was in the Gulf war of 1991, what we know about depleted uranium's effects on human health did not begin there. A sizeable store of knowledge has been gathered over the past 50 years in studies of uranium miners, millers, and other processors worldwide. Two recent reviews of uranium exposure and cancer risk address overall cancer mortality and also lung, lymphoid, and bone cancer, those most likely to be related to internal uranium exposure. The first, by the US Centers for Disease Control and Prevention/Agency for Toxic Substances and Disease Registry, concluded that "no significant differences in cancer [of the lungs] was found between workers who are occupationally exposed to uranium and control populations."[2] A review of over 11 studies in uranium miners attributed an observed increase in lung cancer to radon and its progeny and not to uranium. "There is no unequivocal evidence that inhalation, oral or dermal exposure induces cancers in humans."[6 7] Confounding exposures, often to more radioactive materials, were cited. Long term animal studies with both natural and enriched uranium had negative (nine studies) or equivocal (three studies) results for carcinogenicity.[2]

A second recent review of health effects of uranium authored by the US National Academy of Sciences Institutes of Medicine evaluated existing epidemiological studies more rigorously and gave relative weight to the studies' strengths and weaknesses in their assessments. Regarding the lung cancer risk, "the Committee concludes that there is limited/suggestive evidence of no association between exposure to uranium and lung cancer at cumulative internal dose levels lower than 200 mSv or 25 cGy."[3] This roughly corresponds to the burden occurring from a full year's exposure to a dusty indoor uranium workshop environment.[8]

For both lymphatic and bone cancer the committee concluded that there was inadequate or insufficient evidence to determine whether an association does or does not exist with uranium exposure. Most of the studies cited did not show an excess, but there was also inadequate evidence to dismiss the possibility.[2]

Other evidence comes from a small surveillance study of (then 30 and now 60) US Gulf war veterans who were victims of friendly fire with depleted uranium. About 15 of these veterans possess retained metal fragments of depleted uranium in soft tissue and are excreting raised uranium concentrations in their urine. None of these veterans has leukaemia, bone cancer, or lung cancer.[9] Thus, the argument for uranium being the cause of leukaemia in peacekeeping forces is thin, notwithstanding the short latencies implied, even by the standards of haematological malignancies.

The questions raised as to the connection between cancer and depleted uranium are understandable, however. Having lived through the cold war and beyond, we have all been sensitised to the fear of nuclear exchange, and justifiably so. In addition, many of us, including those in the medical community, have little familiarity with the dose-response curves of health effects caused by radiation exposure. Experts in risk communication tell us that lack of familiarity with a hazard heightens the public perception of risk.[10] In this context pronouncements about background rates of disease observable in populations being statistically expected fail.

Certainly, attribution of disease to "expected" or background levels should be a diagnosis of exclusion, and vigilance is warranted. However, the information we have on cancer risk is not simply a theoretical calculation, as some critics have suggested. Rather, there is both a context in which to evaluate the evidence and a hierarchy of relative weight to assign to types of evidence available for human risk assessment. Heading that list are epidemiological studies of human health effects, which do exist in this case.[3 6 7]

Regarding non-cancer health effects, and references made to Gulf war illness, there is still no single candidate hazard which serves as its unifying explanation, depleted uranium included. Indeed, Gulf war illnesses is a more appropriate title for the groupings of symptoms made by returning US veterans and recalled in the "Balkan syndrome" of affected peacekeepers. The report into Gulf war syndrome of the presidential advisory panel in 1996 stated that there was no evidence of a connection between depleted uranium and Gulf war illnesses.[11]

Some final prevention points still must be made. Strident efforts at hazard communication training are in order for all serving in affected areas in any role—military or humanitarian. Similar efforts must be undertaken for the affected local populations. Low radiation risk issues aside, children should not be playing with depleted uranium penetrators and environmental monitoring (of drinking water, for example) is appropriate in highly affected areas—at the very least for assurance purposes.

It is uncommon to have the benefit of 50 years of human epidemiological evidence in managing any of the environmental or occupational public health problems facing the global medical community today. We should use that evidence in informing the public about their potential health risk and, more positively, in guiding our prevention activities.

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